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DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE

Food and Drug Administration

■

POISONOUS OR DELETERIOUS SUBSTANCES IN PEANUTS, EVAPORATED MILK, FISH AND SHELLFISH

Proposed Rules

DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE

Food and Drug Administration

[21 CFR Part 122]

ACTION LEVEL FOR MERCURY IN FISH AND SHELLFISH

Notice of Proposed Rule Making

Elsewhere in this issue of the FEDERAL REGISTER, the Commissioner of Food and Drugs is issuing proposed regulations concerning the control of poisonous or deleterious substances in food. In accordance with those proposed procedures and principles, the Commissioner is issuing a proposal for an action level for mercury in fish and shellfish.

Most of man's exposure to mercury arises from the contamination of food and possibly drinking water. The levels of mercury found in water treated for drinking are lower than those found in food. Moreover, mercury in water is found primarily in the less toxic inorganic form. Except in occupational exposure, the contribution of inhaled mercury is insignificant in relation to intake from food. The amount of methylmercury (the most toxic organic form of mercury) found in fruits, vegetables, grains, meat and dairy products is very small (reference 4). Of all the commodities analyzed for mercury by the Food and Drug Administration (FDA) in a survey of 10 basic foods and in the food commodity classes of the FDA total diet study, fish was the only commodity shown to present a potential hazard to man (reference 5). Other data show levels of mercury in shellfish (mollusks and crustaceans) similar to the levels in some vertebrate fish (reference 10). The total human intake of mercury from food sources other than fish and shellfish is insignificant.

All forms of mercury are toxic, but it has been established that the form of mercury in edible fish muscle is almost completely methylmercury (reference 1, chapter 3), which is more toxic chronically than other forms of mercury. Currently only limited data are available on the form of mercury in shellfish. These data do indicate, however, that the mercury in shrimp and lobster is largely methylmercury (reference 11). No data are available on the methylmercury concentrations in mollusks. There is no analytical procedure suitable for regulatory use that is capable of measuring levels of methylmercury. Therefore, the action level is expressed in terms of mercury. Since data are not available to establish with certainty the proportion of mercury that is methylmercury in each species of fish and shellfish, all mercury must be regarded as the most hazardous form, methylmercury.

Methylmercury and other forms of mercury serve no essential function in fish and shellfish. They are not normal inherent constituents, but are found in aquatic species because of environmental, industrial, and agricultural contamination. The source of methylmercury contamination in fish and shellfish is mercury contamination of the waters. The mercury that contaminates the aquatic environment has come from two principal sources, leaching or volatilization from natural geological sources and human activities. Significant amounts of mercury enter the environment from man's agricultural and industrial use (chlor-alkali industry, mercury-based slimicides, mining operations, burning of fossil fuels, etc.).

All forms of mercury entering the aquatic environment, whether as a result of man's activities or from natural geologic sources, may be converted by microorganisms in bottom sediments to methylmercury, which can be concentrated by fish and other aquatic species.

Studies by Finnish scientists have shown that methylmercury is lost from fish at an extremely slow rate (reference 2). The loss appears to occur in two stages, first a rapid loss when mercury is being distributed throughout the tissues, a process that lasts a few weeks, and then a very slow loss from the established binding sites. The half-life estimated for this component is approximately 2 years. This extremely slow loss is one of the reasons why fish are a major source of mercury for man. Moreover, during this period the fish are continuously supplied with methylmercury from the water, providing a mechanism for the continuous increase of mercury residues.

Mercury is present in fish and shellfish because of contamination. It is, therefore, an added poisonous or deleterious substance within the meaning of the Federal Food, Drug, and Cosmetic Act. The meaning of that term is discussed in detail in the preamble to the proposed procedures and principles for the control of poisonous or deleterious substances published elsewhere in this issue of the FEDERAL REGISTER.

Mercury contamination of fish and shellfish is unavoidable. Though further addition of mercury to the aquatic environment from industrial sources can be prevented or at least reduced, at present there are no means of removing the mercury from these waters. Moreover, there is no method of processing fish or shellfish so as to remove or reduce the mercury contamination. Control over mercury intake by consumers of fish and shellfish can be achieved only through regulation of the mercury content of the products sold.

Methylmercury poisoning may lead to progressive blindness, deafness, incoordination, intellectual deterioration, and death. The amount of mercury accumulating in the body is directly related to the methylmercury dose size, and a critical concentration of mercury in brain tissues is required to produce neurological symptoms. Because it requires some time for the brain tissue of exposed individuals to reach such levels, due to the time of passage through the blood-brain barrier and redistribution of mercury in the brain, neurological symptoms are not observed during the early periods of exposure, but may develop several weeks after the initial exposure. However, it has been established that toxic concentrations of methylmercury in the brain vary very little between different animals. In all mammals, concentrations of 5 to 10 micrograms per gram (mcg/g) of brain tissue may be associated with neurological symptoms.

At present, there are no sensitive diagnostic techniques to identify possible sub-clinical and latent mercury toxicity. Of particular concern in relation to possible undetected injury are reports that individuals who have been exposed to methylmercury (or other alkyl mercurials) may continue to function in an apparently normal manner and then at death, some years later, show the complete pathologic brain lesions associated with the exposure. It is not known to what extent man's brain can compensate for neuronal loss that may occur following exposure or how neuronal loss following low levels of exposure affects the normal aging process of the brain (reference 3).

Methylmercury readily crosses the placental barrier in pregnant women and enters the fetus (references 1 and 2). It may concentrate in the central nervous system of the developing organism resulting in serious brain damage to the unborn child. Fetal damage may occur at exposure levels lower than that required to produce observable effects in the mother. Therefore, chronic exposure to fish and shellfish containing methylmercury poses a greater potential for danger to women of child-bearing age than to the general population.

Because of these uncertainties about the effects of low levels of methylmercury, the approach to tolerating mercury contamination must generally be cautious.

The potentially hazardous nature of mercury residues in fish and shellfish has been emphasized by the Minamata Bay and Niigata, Japan, poisoning episodes which involved fatalities associated with consumption of contaminated fish and shellfish. Detailed studies of the patients affected during these poisoning episodes, particularly information on mercury levels in blood, hair, and in some cases brain, have provided valuable information for estimating tolerable levels of exposure of man to methylmercury. Additional information has also been provided by (1) studies in Scandinavia on the metabolism of trace amounts of Hg^{203}-labeled methylmercury by man, which have permitted a calculation of the biological half-life of mercury in man (about 70 days) and the relative distribution of mercury in the various parts of the body and (2) the relationship between ingestion of methylmercury from contaminated fish and mercury levels in blood and hair (reference 2).

The 0.5 ppm action level for mercury in fish and shellfish established by the Food and Drug Administration in 1969 incorporated all the significant aspects of the mercury-in-fish problem known at

the time of its establishment. The information available at that time included:

(1) Estimates of levels of intake of contaminated fish and shellfish which resulted in the Japanese poisoning episodes.

(2) Data on the relation of intake of methylmercury to body levels of methylmercury. Some data related intake of methylmercury in man from contaminated fish to levels of mercury in blood. Other human data were from studies on distribution and excretion of raidoisotope-labeled methylmercury in a few volunteers. Data on brain levels of mercury were from test animals and human autopsy cases.

In 1970, a study group of 10 scientists (both FDA and non-FDA) concluded on the basis of their experience in Sweden and Finland, where there had been a great deal of investigation regarding mercury hazards, that the FDA action level of 0.5 ppm was a sound basis for the protection of the public health (reference 2). This conclusion was based in part on data obtained by the Swedish Commission on Evaluating the Toxicity of Mercury in Fish.

The Swedish Commission set forth its recommendations relating to allowable intakes of methylmercury (reference 1). Their recommended tolerable daily intake was based on a set of values derived from data on Swedish individuals, their blood levels of methylmercury, and their dietary intake of fish, as well as the Japanese data, as follows: The lowest whole blood concentration at which toxic symptoms occurred in the Japanese poisoning episodes was estimated to be 0.2 microgram per gram (mcg/g). Swedish data on intake of contaminated fish showed that these blood levels could be reached with a daily intake of 0.3 milligram (mg) by a 70 kilogram (kg) man. Extrapolating from these human data and applying a safety factor of 10 (the usual safety factor applied when human data are used), the acceptable level for whole blood would be 0.02 mcg/g, which would correspond to a daily intake of 0.03 mg by a 70 kg man or 0.4 mcg/kg of body weight. If fish containing 0.5 ppm mercury were eaten daily, the acceptable limit (0.03 mg) would be reached by the daily consumption of 60 grams of fish.

The action level for mercury in fish and shellfish was again reviewed in April 1971 by an ad hoc committee of scientific and medical experts from this country and Canada with respect to the high levels of mercury found in swordfish. The committee supported maintaining the 0.5 ppm guideline (reference 8).

The most recent data on the toxicity of methylmercury are from a study of a widespread outbreak of methylmercury poisoning in Iraq in 1972 in which farmers and their families were exposed to methylmercury when they ate homemade bread prepared from seed wheat treated with a methylmercurial fungicide (reference 9). The amount of mercury in the body at the onset of toxic symptoms (threshold body burden) was calculated to be 25 to 40 mg. This agrees remarkably well with a threshold body burden of 20 to 40 mg of mercury computed by the Swedish Commission from the whole blood concentrations at onset of symptoms reported from Japanese data (reference 1). While this is a preliminary interpretation of the Iraqi data, it does tend to confirm the Swedish Commission's allowable intake level, for which a maximum permissible level of 0.5 ppm will provide adequate protection.

Because this important Iraqi study is incomplete and other mercury studies are in progress, the Commissioner concludes that it would be inappropriate to set a formal tolerance at this time. When the study of the Iraqi poisoning episode is fully developed and published, the Commissioner will reevaluate it and all other available information and consider whether to establish a formal tolerance for mercury in fish and shellfish. The Commissioner concludes that in the interim, it is appropriate to issue an action level for mercury in fish and shellfish as a regulation.

As demonstrated above, an action level of 0.5 ppm permits a daily consumption of 60 grams of fish and shellfish contaminated to that extent, while still maintaining a tenfold margin of safety over the level of mercury intake known to be toxic. The 60 g/day figure is based on a mercury concentration of 0.5 ppm in all fish and shellfish consumed, but because actual concentrations are much less in most fish and shellfish (references 4, 6 and 10), there is an additional safety margin. In addition, the average consumption of fish and shellfish in this country is regarded as considerably less than 60 grams per day (reference 1, chapter 13; references 6 and 12).

A National Marine Fisheries Service (NMFS) survey determined that only 1.8 percent of the population consumes an average of more than 60 grams of fish and shellfish per day (reference 7). The vast majority of the population is therefore protected by the tenfold margin of safety. Additional protection is provided by the fact that actual concentrations in most fish and shellfish are below 0.5 ppm mercury and the fact that consumption is less than an average of 60 grams per day.

It is not possible, however, to provide this same high level of protection to every person without excluding a great amount of fish and shellfish from the market. The NMFS survey indicated that 1 percent of the participants in the survey consumed an average of 77 grams daily and 0.1 percent consumed 165 grams daily. At the very high consumption of 165 grams per day, the tenfold margin of safety is reduced to less than four; although, it may be increased above that figure by the additional margin resulting from consumption of fish and shellfish with less contamination than 0.5 ppm mercury.

An action level of about 0.18 ppm would be necessary to assure a tenfold margin of safety for those individuals who consume approximately 165 grams of fish and shellfish per day. Since the mean mercury levels for certain species of fish, e.g., tuna and red snapper, exceed 0.18 ppm and a significant portion of other species also exceeds that level (references 5 and 6), an inordinate amount of fish would be excluded from the market by such an action level.

The number of fish and shellfish that would be excluded from sale at a given action level is a reflection of the degree to which the contaminant is unavoidable. In establishing an action level, the Commissioner must consider the extent to which the contaminant is unavoidable, which is the policy embodied in section 406 of the act.

In balancing the unavoidability of mercury in fish and shellfish against the need for protection of the public health, the Commissioner concludes that it would be inappropriate to exclude a vast amount of fish and shellfish from the market in order to provide a large margin of safety for those who consume far more than the average person. The Commissioner notes that a person would have to consume every day the extraordinarily high amount of 600 grams of fish and/or shellfish contaminated at the full 0.5 ppm level to reach the blood levels where symptoms have been known to occur. Thus some margin of safety is still provided for even the largest consumers of these foods.

In setting a tolerance or action level, the Commissioner considers the other ways in which the consumer may be affected by the same substance or other substances. As noted above, fish and shellfish are the major sources of dietary mercury, and the presence of mercury in other foods is too insignificant to affect the action level for mercury in fish and shellfish. The Commissioner has also considered the effects mercury has on human health in combination with other substances, including other heavy metals. Information is not available that demonstrates that the action level for mercury in fish and shellfish should be affected by the presence of other substances in food.

As stated above, this action level is based on the assumption that all mercury detected in fish and shellfish is methylmercury, since there is no suitable regulatory test for methylmercury and since the relationship of the amount of methylmercury to the amount of total mercury cannot be established with certainty for every species. The Commissioner will consider higher action levels for particular species or categories of fish and shellfish if adequate data are presented to establish that the consistent proportion of mercury that is methylmercury is relatively low.

The regulations also prescribes sampling and analytical methods by which compliance with the proposed limitation will be determined.

Taking into account (1) that mercury contamination of the aquatic habitat and thereby of fish and shellfish is ubiquitous and unavoidable, (2) that mercury cannot be removed from fish and shellfish tissue during processing,

(3) the extent to which mercury in fish and shellfish is unavoidable, (4) toxicological data on the effects of mercury, and (5) the other ways in which the consumer may be affected by mercury and other substances, the Commissioner proposes to establish an action level for mercury in fish and shellfish. Accordingly, an action level of 0.50 ppm is proposed.

REFERENCES

A copy of each reference document is on file with the Hearing Clerk, Food and Drug Administration, Rm. 4–65, 5600 Fishers Lane, Rockville, MD 20852, and may be seen during working hours, Monday through Friday.

1. "Methyl Mercury in Fish; A Toxicologic-Epidemiologic Evaluation of Risks. Report from an expert group." Nordisk Hygienisk Tidskrift, Supp. 4, 1971.

2. "Hazards of Mercury," Special Report to the Secretary's Pesticide Advisory Committee, Department of Health, Education, and Welfare, November 1970, Environmental Research 4:1–69, 1971.

3. Shibko, S. I., Testimony before Southwestern South Dakota Enforcement Conference, Rapid City, SD, October 19–21, 1971.

4. "Evaluation of Certain Food Additives and the Contaminants Mercury, Lead, and Cadmium," Sixteenth Report of the Joint FAO/WHO Expert Committee of Food Additives.

5. Simpson, R. E., Horwitz, W. and Roy C. A., "Residues in Food and Feed, Survey of Mercury Levels in Fish and Other Foods," Pesticides Monitoring Journal 7, No. 3/4, 127–138 (1974).

6. Analysis of Data from Mercury-Wholesale Fish Survey, Division of Mathematics, Bureau of Foods, Food and Drug Administration, June 1971.

7. Finch, R., "Effects of Regulatory Guidelines on the Intake of Mercury from Fish—The MECCA Project," Fishery Bulletin 71(3): 615, 1973.

8. HEW News, 71–25, Thursday, May 6, 1971.

9. Bakir, F., Clarkson, T. W., et al., "Methylmercury Poisoning in Iraq," Science, pp. 230–241, July 20, 1973.

10. Memorandum: Proposed 0.5 ppm Tolerance for Mercury in Fish; Shellfish Sanitation Branch to Division of Regulatory Guidance, November 12, 1974.

11. Letter: Clarkson to Shibko, July 12, 1972.

12. Compilation of Data on Consumption of Fish and Shellfish in U.S., Guidelines and Compliance Research Branch, November 14, 1974.

Therefore, pursuant to provisions of the Federal Food, Drug, and Cosmetic Act (secs. 306, 402, 406, 701(a), 52 Stat. 1045–1046 as amended, 1049, 1055; 21 U.S.C. 336, 342, 346, 371(a)) and under authority delegated to him (21 CFR 2.120), the Commissioner proposes that Part 122 be amended by adding thereto a new Subpart C consisting at this time of § 122.200 as follows:

Subpart C—Action Levels for Added Poisonous or Deleterious Substances

§ 122.200 Mercury in fish and shellfish.

(a) An action level of 0.50 part per million is established for mercury in fish and shellfish (mollusks and crustaceans), both raw and processed.

(b) Compliance with this section shall be determined as follows:

(1) (i) For processed fish and shellfish, a lot shall consist of a collection of primary containers or units of the same size, type, and style produced under conditions as nearly uniform as possible, designated by a common container code or marking, or, in the absence of any common container code or marking, a day's production.

(ii) For unprocessed fish and shellfish (raw, fresh, or frozen, usually whole), a lot shall consist of a collection of fish or shellfish from a shipment or storage designation, of a uniform size and kind (or species) taken from similar waters of origin. Where the waters of origin are not known, fish or shellfish of the same size and kind will be considered to comprise a lot.

(2) The sample for mercury analysis shall consist of a composite of the edible portion of 12 subsamples (primary packages or units from a lot) chosen randomly to be representative of the lot. The edible portion of vertebrate fish excludes heads, scales, tails, fins, viscera, and inedible bones.

(3) Composites shall be analyzed by the official procedure of the Association of Official Analytical Chemists, described in "Changes in Methods: 25.A01–25.A03," Journal of the Association of Official Analytical Chemists (JAOAC), Vol. 54, p. 466 (1971).[1]

(4) A lot does not comply with the action level established in paragraph (a) of this section if the value of the analysis of the composite of randomly chosen subsamples is greater than 0.50 ppm mercury.

Interested persons may, on or before March 6, 1975, file with the Hearing Clerk, Food and Drug Administration, Rm. 4–65, 5600 Fishers Lane, Rockville, MD 20852, written comments (preferably in quintuplicate) regarding this proposal. Received comments may be seen in the above office during working hours, Monday through Friday.

Dated: November 29, 1974.

A. M. SCHMIDT,
Commissioner of Food and Drugs.

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[21 CFR Part 122]

LEAD IN EVAPORATED MILK AND EVAPORATED SKIM MILK

Proposed Tolerance

Elsewhere in this issue of the FEDERAL REGISTER, the Commissioner of Food and Drugs is issuing proposed procedural regulations concerning the control of poisonous or deleterious substances in food. In accordance with those proposed procedures and principles, the Commissioner is issuing a proposal for a tolerance for lead in evaporated milk and evaporated skim milk.

Early in 1972, the Food and Drug Administration conducted investigations and examinations of four evaporated milk manufacturing plants in different geographical areas of the country to determine baseline values of lead in raw milk and evaporated milks (evaporated milk and evaporated skim milk). The following information was obtained from these investigations (reference 1): (1) Samples of raw milk were found to contain from none detected to 0.28 part per million (ppm) lead with an average of 0.09 ppm and (2) levels of lead in evaporated milks ranged from 0.21 ppm to 1.10 ppm with an average of 0.52 ppm.

These results were supported by other investigations published in the scientific literature (references 2 and 3) which revealed ranges of 0.02 to 0.08 ppm and 0.01 and 0.21 ppm lead, each with averages of 0.05 ppm, for raw milk. Values were reported for lead in evaporated milk ranging from 0.09 to 0.72 ppm with an average of 0.35 ppm.

The levels of lead in evaporated milks were higher than would be caused solely by the normal procedure of concentrating raw milk 2 to 2.5 times during the evaporation process, and the Commissioner concluded from these data that a substantial portion of the lead in evaporated milk was being added during, or as a result of, processing.

Lead in evaporated milk is, therefore, an added poisonous or deleterious substance within the meaning of the Federal Food, Drug, and Cosmetic Act. The meaning of that term is discussed in detail in the preamble to the proposed procedures and principles for the control of poisonous or deleterious substances published elsewhere in this issue of the FEDERAL REGISTER.

The evaporated milk industry was informed of the seriousness of the problem of added lead in their product and of the need to develop a solution. In February 1972, the Evaporated Milk Association (EMA) formed a task force composed of representatives of evaporated milk manufacturers and started a series of investigations (reference 4). As a result of these studies, some modifications in can manufacturing procedures were made, and the manufacturing companies set up processing lines using the new techniques in the effort to reduce the lead levels in evaporated milk and evaporated skim milk.

The evaporated milk industry also began a monitoring and sampling program in January 1973 (reference 4), to provide assurance that evaporated milk and evaporated skim milk entering consumer distribution channels would not contain more lead than the amount which is unavoidable using the new technology. This monitoring program requires that all plants producing evaporated milk or evaporated skim milk for retail sale monitor the finished product.

The levels of lead actually present in raw milk were probably significantly lower than the approximately 0.09 ppm reported, based on investigations prior to

[1] Copies may be obtained from: Association of Official Analytical Chemists, P.O. Box 540, Benjamin Franklin Station, Washington, DC 20044.

1973. Improvements in analytical methodology that have been developed since 1973 cast some doubt on the validity of the earlier data. Using the improved methodology, an FDA survey of whole milk in fiscal year 1974 (July 1973–June 1974) resulted in data in which 0.021 ppm was the average amount of lead determined in 60 samples. The lead findings ranged from none detected to 0.069 ppm (reference 5). More samples would have to be analyzed to obtain a truly reliable measure of the average lead content of all whole milk, but it be concluded that it is significantly less than the 0.09 ppm reported from earlier data.

While the same analytical problems existed for evaporated milk as for raw or whole milk, those problems were not as significant at the levels of lead that occurred in the evaporated product. The 0.09 ppm average lead levels found in whole milk were at the limit of sensitivity of the analytical methods employed, where results are generally less precise.

Data reported to FDA from the industry monitoring program for the first half of 1973 showed an average of 0.12 ppm lead in over 3,000 samples of evaporated milk and evaporated skim milk. The results ranged from 0.01 ppm to 0.46 ppm (reference 6). These data are generally consistent with the results of a survey conducted by FDA in 1973 in which 83 samples of evaporated milk were found to contain an average of 0.12 ppm lead, with a range of 0.02 to 0.37 ppm (reference 7). Results from an independent study also were in agreement: 15 samples of evaporated milk contained an average of 0.11 ppm lead with a range of 0.04 to 0.22 ppm and 11 samples of evaporated skim milk averaged 0.06 ppm lead with a range of 0.04 to 0.07 ppm (reference 8).

Industry data for the second half of 1973 and the first 4 months of 1974, in which 3,015 samples contained an average of 0.12 ppm lead with a range of 0.02 to 0.33 ppm (reference 9), indicate that the changes in technology have resulted in the reduction of lead levels in evaporated milk and evaporated skim milk to a stable level which should not exceed 0.3 ppm.

Clinically overt pediatric lead toxicity involves damage to the central nervous system and the kidneys as well as an impairment in the production of blood cells. The central nervous system effects are principally responsible for the morbidity and mortality of the disease (reference 10). While the incidence of fatality due to coma and convulsions produced by lead ingestion has declined markedly over the past 20 years due to both the availability of methods for the earlier detection of the disease and the use of chelation therapy, it has been established that less severe lead toxicity may still result in impairment of the central nervous system. Subtle central nervous system damage that has been associated with lead includes hyperactivity (references 11 and 12) and impaired motor coordination (references 13 and 14).

The pathological changes in central nervous system function frequently remain even though it is possible to reduce the body burden of lead with chelation therapy. Perlstein and Attala (reference 15) found that 39 percent of children treated by chelation therapy showed neurologic sequelae including mental retardation, recurrent seizures, cerebral palsy, and optic atrophy. Chisolm (reference 16) has also reported persistent neurologic deficits in children after chelation therapy treatment for lead poisoning. Thus, it is clear that while the biochemical changes due to lead may be reversed by removal of lead, neurological changes may remain for prolonged periods of time.

When there is an increased body burden of lead, it is reflected by an increased lead concentration in the blood. A blood lead concentration greater than 40 micrograms (mcg) per 100 milliliters (ml) of whole blood is considered evidence of undue exposure to lead (reference 17). The incidence of blood lead concentrations in excess of 40 mcg/100 ml of whole blood varies with the populations studied. Generally, the frequency is highest among children living in center-city areas with poor housing. In such center-city areas, 25 to 45 percent of young children show evidence of undue exposure to lead (reference 18). The problem is not limited, however, to large urban areas since the incidence of blood lead concentrations greater than 40 mcg/100 ml has been reported to vary between 9.6 and 31.3 percent in Illinois cities under 100,000 population (reference 19).

Severe lead poisoning is almost invariably associated with pica for lead containing substances such as paint, plaster, newspaper, or dirt. Children with symptomatic lead poisoning often have blood lead concentrations greater than 60 mcg/100 ml of whole blood. Subtle neurological changes associated with lead are thought to occur at blood lead concentrations of 40 to 60 mcg/100 ml of whole blood although this is not fully established (references 11, 12, 13, 14). While the majority of children with blood lead concentrations greater than 40 mcg/100 ml of whole blood have pica, many do not have a demonstrable pica-related source of lead.

Nonpica sources of lead include food, water, and air. While the relative magnitude of these sources varies in different environments, food is generally the largest contributor to lead exposure in nonpica children. The average total dietary intake of lead of a 2-year old child has been estimated to be approximately 75 mcg per day, with about 45 mcg contributed by the dairy products that constitute about 36 percent of this average diet. Similarly, the estimated average intake of a 6-month old infant who consumes only infant foods would be approximately 120 mcg per day, with about 70 mcg contributed by the dairy products that constitute 59 percent of the average diet (reference 20).

Lead metabolism and the metabolic effects of various quantities of lead have been studied in adults (reference 21). Similar experimental data are not available for children; however, some knowledge of the quantities of lead which will produce various metabolic effects has been obtained (reference 22). An ad hoc committee of experts in pediatric lead toxicity, appointed by the U.S. Department of Health, Education and Welfare, has suggested that 300 mcg of elemental lead per day be considered the maximum daily permissible intake from all sources for children between 1 and 3 years of age (reference 22). Ingestion of lead in excess of this amount results in an increased body burden of lead and the beginning of lead-induced metabolic changes, specifically impaired heme synthesis. The World Health Organization has established a provisional tolerable weekly lead intake for adults at 3 milligrams of lead per person or 0.05 milligram/kilogram body weight (reference 23). No provisional allowances for lead have yet been established for infants or children; although on the basis of a lesser body weight, the total tolerable intake would most likely be smaller for children.

It is thought that the young have greater susceptibility to the toxic effects of lead. Although experimental data on this point are limited, there is recent evidence of increased toxicity and a greater retention of ingested lead in young as compared to adult animals (reference 24). It has previously been shown that very young animals absorb a greater portion of ingested lead than do adult animals (references 25 and 26). Children have been shown to absorb and retain substantially larger percentages of dietary lead than do adults (reference 27).

Although evidence of greater lead susceptibility of children when compared to adults has not been firmly established, it is evident that children, as a result of a higher caloric requirement due to their higher metabolic rate and a consequently more rapid respiratory rate, have a greater degree of lead exposure per kilogram of body weight.

The lead concentration of evaporated milk is of particular interest in infant feeding. Formula made from evaporated milk often provides a high percentage of the calories ingested by infants from birth to 6 months of age. Between 6 months and 1 year, formula frequently provides at least half of the caloric intake. In addition, the high milk diet of infants presents special problems because of the low iron intake which accompanies these diets if they are unsupplemented with iron. It has been shown experimentally that low iron intake will increase susceptibility to lead toxicity (reference 28), and that the retention of lead by animals fed milk diets is unusually high (reference 29).

The Commissioner has determined that the proposed 0.3 ppm lead tolerance will provide adequate protection for infants and young children from ingestion of hazardous levels of lead from evaporated milk and evaporated skim milk. If all evaporated milks were to contain the full 0.3 ppm lead, the calculated daily lead intake for infants would range from

about 70 mcg for infants from birth to 3 months, who obtain 80 to 90 percent of their total calorie intake from formula, to about 170 mcg for 9 to 12 month olds, who obtain 80 percent of their calorie intake from formula (reference 30). These lead intake levels are significantly lower than the 300 mcg allowable daily intake for children and, though an allowable level for infants has not been determined, such a level would probably not be exceeded by users of evaporated milks containing 0.3 ppm lead.

Based upon an analysis of currently available data, a level of 0.3 ppm lead is expected to occur only occasionally (reference 9). In a lot with an average lead level less than 0.3 ppm, most individual containers will have less than 0.3 ppm. With a maximum permissible level of 0.3 ppm lead in a 12-can composite sample, lots averaging more than 0.3 ppm should be rejected. Thus, the possibility of any individual's being exposed to the calculated intake of 170 mcg lead per day over an extended period is remote.

The limitation on lead in evaporated milk and evaporated skim milk is proposed to be a tolerance. Limiting an unavoidable substance through use of a tolerance rather than an action level is appropriate when there are no changes foreseeable in the near future that might affect the appropriateness of the limitation established. The Commissioner concludes that additional improvements in can technology, further reducing the levels of lead in evaporated milk and evaporated skim milk, are not expected in the near future. In addition, the scientific evidence on the toxicity of lead is sufficiently well established that no data are expected to be developed in the near future that would cause the Commissioner to alter the limitation proposed.

In setting a tolerance, the Commissioner considers the other ways in which the consumer may be affected by the same substance or by other substances. As noted above, dairy products are a major source of dietary lead, and the presence of lead in other foods consumed by infants and young children is not significant enough to affect the tolerance for lead in evaporated milk and evaporated skim milk.

One potential exposure to lead, which is not accountable by current food intake data (reference 5), is leachable lead from pottery (ceramics) and enamelware suitable for liquid food use. Since the manner and extent to which these articles are used for liquid food in the home has not been documented, the Commissioner is not able to establish the exact contribution of this leachable lead to total human dietary intake.

The Commissioner concludes, however, that whatever this contribution may be, it should not, at this time, bear on the tolerance level being proposed for lead in evaporated milk and evaporated skim milk. In part, this tolerance takes into account the extent to which lead in these foods is unavoidable. Thus, rather than having this tolerance any lower, the Commissioner considers it proper and in the public interest to study the present FDA action level of 7 micrograms/milliliter for leachable lead in pottery and enamelware, and at the completion of that study, to determine whether the action level for leachable lead in these articles should be changed or a tolerance established. The proposed tolerance or action level would be based on the criteria contained in the proposed procedures and principles for the control of poisonous or deleterious substances published elsewhere in this issue of the FEDERAL REGISTER.

The Commissioner has also considered the effects lead in combination with other substances, including other heavy metals, has on human health. Information is not available that demonstrates that the tolerance for lead in evaporated milk and evaporated skim milk would be affected by the presence of other substances in food.

The regulation also prescribes sampling and analytical methods by which compliance with the proposed limitation will be determined. Because of the possible variation in the results obtained by the analytical method prescribed, the regulation provides that the average of duplicate analyses shall be used.

Taking into account (1) the extent to which lead is present in raw milk and cannot be removed in the production of evaporated milk and evaporated skim milk; (2) the fact that can technology has been improved so that industry production practices can effectively eliminate the avoidable lead from these products; (3) the upper limit of safety based on present toxicological knowledge; and (4) the ways in which consumers may be affected by lead and other substances, the Commissioner proposes to prescribe a tolerance for lead in evaporated milk and evaporated skim milk, above which such products will be deemed unsafe within the meaning of section 406 of the act. Accordingly, a tolerance of 0.3 ppm is proposed.

REFERENCES

A copy of each reference document is on file with the Hearing Clerk, Food and Drug Administration, Room 4-65, 5600 Fishers Lane, Rockville, MD 20852, and may be seen during working hours, Monday through Friday.

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Therefore, pursuant to provisions of the Federal Food, Drug, and Cosmetic Act

(secs. 306, 402, 406, 701, 52 Stat. 1045–1046 as amended, 1049, 1055–1056 as amended by 70 Stat. 919 and 72 Stat. 948; 21 U.S.C. 336, 342, 346, 371) and under authority delegated to him (21 CFR 2.120), the Commissioner proposes that Part 122 be amended by adding a new § 122.102 as follows:

§ 122.102 Lead in evaporated milk and evaporated skim milk.

(a) A tolerance of 0.3 part per million (ppm) is established for lead in evaporated milk and evaporated skim milk.

(b) Compliance with this section shall be determined as follows:

(1) A lot shall consist of a collection of primary containers or units of the same size, type, and style produced under conditions as nearly uniform as possible, designated by a common container code or marking, or in the absence of common container code or marking, a day's production.

(2) The sample for lead analysis shall consist of a composite of randomly chosen sample units from a lot as follows: 12 cans of the 13 fluid ounce size or 29 cans of the 5½ fluid ounce size, or the equivalent in the case of cans of other sizes.

(3) Composites shall be analyzed in duplicate by the procedures of the Association of Official Analytical Chemists (AOAC): The atomic absorption technique as described in "Official Methods of Analysis of the Association of Official Analytical Chemists," 11th Ed., 3d Supplement, 25.C12–25.C17, or the anodic stripping voltammetry technique as described in the Journal of the AOAC, Vol. 56, p. 1247 (1973).[1]

(4) A lot does not comply with the tolerance established in paragraph (a) of this section if the average value of duplicate analyses of the composite of randomly chosen sample units is more than 0.3 ppm lead.

Interested persons may, on or before March 6, 1975, file with the Hearing Clerk, Food and Drug Administration, Rm. 4–65, 5600 Fishers Lane, Rockville, MD 20852, written comments (preferably in quintuplicate) regarding this proposal. Received comments may be seen in the above office during working hours, Monday through Friday.

Dated: November 29, 1974.

A. M. SCHMIDT,
Commissioner of Food and Drugs.

[FR Doc.74-28406 Filed 12-5-74;8:45 am]

[21 CFR Part 122]

POISONOUS OR DELETERIOUS SUBSTANCES IN FOOD

Notice of Proposed Rule Making

Elsewhere in this issue of the FEDERAL REGISTER the Commissioner of Food and Drugs is issuing proposed regulations concerning permitted levels of mercury in fish and shellfish (mollusks and crustaceans), lead in evaporated milk and evaporated skim milk, and aflatoxin in shelled peanuts and peanut products used as human foods. The Commissioner has concluded that Part 122, promulgated in an order published in the FEDERAL REGISTER of July 6, 1973 (38 FR 18096), should be expanded to deal with all poisonous or deleterious substances in food, including food-contact surfaces, pet food, and animal feed, to implement sections 402(a) and 406 of the Federal Food, Drug, and Cosmetic Act.

Section 402(a)(1) of the act provides that a food shall be deemed to be adulterated:

> If it bears or contains any poisonous or deleterious substance which may render it injurious to health; but in case the substance is not an added substance such food shall not be considered adulterated under this clause if the quantity of such substance in such food does not ordinarily render it injurious to health.

Section 402(a)(2) of the act provides that a food shall be deemed to be adulterated:

> (A) If it bears or contains any added poisonous or added deleterious substance (other than one which is (1) a pesticide chemical in or on a raw agricultural commodity; (ii) a food additive; (iii) a color additive; or (iv) a new animal drug) which is unsafe within the meaning of secton 406, or (B) if it is a raw agricultural commodity and it bears or contains a pesticide chemical which is unsafe within the meaning of section 408(a); or (C) if it is, or it bears or contains, any food additive which is unsafe within the meaning of section 409: *Provided*, That where a pesticide chemical has been used in or on a raw agricultural commodity in conformity with an exemption granted or a tolerance prescribed under section 408 and such raw agricultural commodity has been subjected to processing such as canning, cooking, freezing, dehydrating, or milling, the residue of such pesticide chemical remaining in or on such processed food shall, notwithstanding the provisions of sections 406 and 409, not be deemed unsafe if such residue in or on the raw agricultural commodity has been removed to the extent possible in good manufacturing practice and the concentration of such residue in the processed food when ready to eat is not greater than the tolerance prescribed for the raw agricultural commodity; or (D) if it is, or it bears or contains, a new animal drug (or conversion product thereof) which is unsafe within the meaning of section 512.

Section 406 of the act permits the establishment of tolerances for added poisonous or deleterious susbtances in food; section 408 of the act permits the establishment of tolerances for pesticide chemicals in or on raw agricultural commodities; and section 409 of the act permits the establishment of tolerances for food additives.

The basic provision of the act dealing with poisonous or deleterious substances in food is section 402(a)(1) of the act, which applies to all poisonous or deleterious susbtances, whether they are naturally occurring or added. It is deliberately cumulative with the provisions relating to establishment of tolerances for food substances referenced in section 402(a)(2) of the act and was retained from the Food and Drugs Act of 1906 because its language had been construed by the Supreme Court in United States v. Lexington Mill & Elevator Co., 232 U.S. 399 (1974). See Hearings on S. 2800 Before the Senate Committee on Commerce, 73d Cong., 2d Sess. 530 (1934). Its broad scope is shown by the fact that the related provisions in sections 406, 408(a), 409(a), and 512(k) of the act exempt a substance from section 402(a)(1) only if a regulation has been promulgated under the authority of those other sections.

Section 402(a) of the act distinguishes between naturally occurring and "added" substances and sets different standards for each. Naturally occurring substances are prohibited only if they render the food "ordinarily" injurious to health. Added substances are prohibited if they "may" be injurious to health; if such substances are necessary or unavoidable, special procedures are established by which they can be regulated.

"Added" is a statutory term of art encompassing all ingredients which are not inherent and intrinsic parts of a food. The term "added" was introduced into Federal food law in the drafting of the 1906 act when the U.S. Department of Agriculture (USDA) opposed a provision defining a food product as adulterated if it was injurious to health. The Department noted that such a provision would outlaw coffee, tea, and other foods containing deleterious ingredients as natural constituents. See the testimony of the Commissioner in Hearings on H.R. 6906, H.R. 8805, H.R. 8941, and S. 5 Before A Subcommittee of the House Committee on Interstate and Foreign Commerce, 74th Cong., 1st Sess. 58 (1935). To exempt such foods containing naturally occurring deleterious ingredients, the Senate committee considering the bill in 1906 adopted the word "added." See 40 Cong. Rec. 897 (1906). Although the word chosen implies that the statute is concerned with the act of addition, the legislative history makes clear that the term seeks rather to establish a standard based upon the necessary and inherent normal condition of the food.

Actions of the USDA after enactment of the 1906 act demonstrate that all contamination was regarded as "added." In 1914, the USDA announced that it had been investigating the presence of lead and arsenic in food, noting that such contaminants were "usually introduced into such products through the use of impure raw materials or from the apparatus or utensils employed in the processes of manufacture." The announcement further stated that "this bureau holds that food containing arsenic or lead, added in any manner, is adulterated, in that it contains an added poisonous or deleterious ingredient which may render the product injurious to health." U.S. Department of Agriculture, Bureau of Chemistry, Service and Regulatory Announcements 312–313 dated June 23, 1914.

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[1] Copies may be obtained from: Association of Official Analytical Chemists, P.O. Box 540, Benjamin Franklin Station, Washington, DC 20044.

Throughout the hearings leading to the 1938 act, it remained clear that substance was "added" within the meaning of the statute whenever it was not inherent in nature, and not just when it was purposefully added. Thus, in explaining a proposed provision equivalent to the present section 402(a)(1) of the act, which removed the 1906 act's limitation to added ingredients, the Commissioner testified that the effect of eliminating the reference to added ingredients was to include deleterious ingredients "normal to the food product." Hearings on S. 2800, supra, at 528. The Commissioner's examples of added substances clearly included environmental and industrial contaminants. One example given of food containing an added poisonous substance was sugar contaminated by smoke from a nearby smokestack. Lead pipes, lead manufacturing equipment, and lead containers were also identified as sources of added poisonous substances. Hearings on H.R. 6906, supra, at 59–60; Hearings on S. 1944 Before a Subcommittee of the Senate Commerce Committee, 73d Cong., 2d Sess. 25, 27 (1933). The Commissioner testified at one point that some added poisonous or deleterious substances were "more or less universal," and at another time that "[t]here are very few things * * * in which you will not find contaminating products in some small degree." Hearings on S. 1944, supra, at 18; Hearings on H.R. 6906, supra, at 59. This emphasis on the ubiquity of added poisonous or deleterious substances clearly demonstrates that the term was understood to include all food contaminants.

The legislative history also identifies examples of foods naturally containing poisonous or deleterious substances and thus not subject to the "added" provisions of section 402(a)(1) of the act. These examples are Burma beans, which contain a glucoside that yields prussic or hydrocyanic acid; rhubarb, which contains oxalic acid; and coffee and tea. See Hearings on S. 1944, *supra*, at 17; Hearings on S. 2800, *supra*, at 29; Hearings on H.R. 6906, *supra*, at 58. Except for substances whose deleterious nature is inherent to the natural state of the food, and thus similar in origin to these examples, all poisonous and deleterious components are "added" within the meaning of the act. Moreover, when a naturally occurring poisonous or deleterious substance is increased to abnormal levels through mishandling or other intervening act, it is "added" to the extent of such increase.

Judicial decisions have recognized that the scope of the "added" provisions of the law extends to all poisonous or deleterious substances not inherently a component of the food. In United States v. 1,600,800 Pounds of White Corn, Civil No. T–4173 (D. Kan., Dec. 18, 1970), the court held that aflatoxin in corn was an added poisonous or deleterious substance within the meaning of the act since aflatoxin is not a natural constituent of corn. In *United States* v. *1200 Cans, Pasteurized Whole Eggs*, 339 F. Supp. 131, 136 (N.D. Ga. 1972), the court recognized that salmonella in eggs is an added poisonous or deleterious substance.

Section 406 was included in the 1938 act to permit the establishment of tolerances for added poisonous or deleterious substances which are required in the production of food or otherwise cannot be avoided by good manufacturing practice. A limitation may be placed upon such substances to the extent necessary for the protection of the public health. Although formal tolerances under section 406 have not been used by the Food and Drug Administration, except for the tolerance for PCB's published in the FEDERAL REGISTER of July 6, 1973 (38 FR 18096), informal action levels have frequently been utilized to implement this provision of the law and a number of those action levels exist today. In the past, such action levels have been issued through press releases and other public announcements but have not been published in the FEDERAL REGISTER or codified as regulations.

Section 408 was added to the act in 1954 to permit the establishment of tolerances for pesticide chemicals in or on raw agricultural commodities. This provision of the law was administered by the Food and Drug Administration (FDA) until the establishment of the Environmental Protection Agency (EPA) in 1970.

In 1958, Congress enacted the Food Additives Amendment, which affected the legal status of some of the contaminants previously regulated only as added poisonous or deleterious substances. The legislation was basically in two parts: first, a broad definition of "food additive" and a general prohibition of food additives not demonstrated to be safe; and second, a procedure by which the use of safe food additives could be approved. This two-part approach was the congressional answer to what was identified as a "dual problem." See H.R. Rep. 2284, 85th Cong. 2d Sess. 1 (1958). The first part of the problem was that under section 402(a)(1) and (2)(A) of the act, the burden is on the Government to show that a food substance is poisonous or deleterious. As the House Commerce Committee noted, "to prove an untested substance poisonous or deleterious may require approximately 2 years or more of laboratory experiments with small animals and during this period the Government cannot prevent the use of such a substance in food." The amendment therefore shifted the burden of proof in enforcement actions brought against food containing poisonous or deleterious substances to the food manufacturer.

The definition of "food additive" established in section 201(s) of the act is not limited to intentional additives. The breadth of the language in section 201(s) of the act includes any food substance, and excludes only those substances which cannot reasonably be expected to become a component of food. The statutory definition thus includes what are referred to as "incidental additives" as well as "intentional additives". See H.R. Rep. No. 2284, supra, at 3. Any added poisonous or deleterious substance in food, unless accidental and unforeseeable or specifically exempted by section 201(s) of the act, is a food additive. A food naturally containing a poisonous or deleterious substance becomes a food additive if it is not generally recognized as safe and is added to other food or undergoes processing. Food-packaging material may also contain food additives. The broad scope of section 201(s) of the act has been judicially recognized in United States v. Ewig Bros. & Co., Nos. 73–1008, 73–1454 (7th Cir., Aug. 28, 1974), and United States v. City Smoked Fish Co., Civil No. 33989 (E.D. Mich. 1970, 1972) in which DDT in smoked fish was held to be a food additive. The court in United States v. Articles of Food, 370 F. Supp. 371 (E.D. Mich. 1974), held that substances migrating from pottery plates are food additives.

The Food Additives Amendment of 1958 was also intended to rectify the second part of the dual problem which was that "present law entirely prohibits the use of these additives even if their use at safe levels would advance our food technology and increase and improve our food supplies." H.R. Rep. 2284, supra, at 1–2. Section 409 of the act was enacted to permit issuing a regulation prescribing the conditions under which an additive may be safely used. Before any such regulation can be issued, the statute requires that the safety of the food additive be demonstrated. The statute also requires that the food additive accomplish a physical or other technical effect and requires the regulation to restrict use of the food additive to the lowest level necessary for that purpose.

When the Food Additives Amendment of 1958 was enacted, the provisions of section 406 of the act were not repealed. Although all added poisonous or deleterious ingredients are food additives, except when they appear in food accidentally and unforeseeably or are exempted under section 201(s) of the act because they are otherwise regulated under the act, the tolerance-setting provisions of section 406 of the act were left intact to deal with those unavoidably added poisonous or deleterious ingredients that could not meet the high standards for issuance of a regulation under the authority of section 409 of the act. A number of added poisonous or deleterious substances, which are also food additives within the meaning of section 201(s) of the act, are unavoidable but cannot meet the requirements for a section 409 regulation because their safety cannot be demonstrated and because they serve no functional purpose. A prominent example is lead, which was one of the contaminants most frequently mentioned in the legislative history of the 1938 act and one of the prime contaminants with which section 406 was enacted to deal. Lead cannot be the subject of a food additive regulation under section 409 of the act even at trivial levels because it serves no functional purpose. Section 406 of the act, therefore, remains in force to control the use of such substances, since there would otherwise be no statutory means available to recognize

their unavoidability and to exercise reasonable control over their presence.

Thus, there is nothing in the language of section 402(a)(2)(A) of the act to prevent issuance of a regulation under section 406 of the act for an added poisonous or deleterious substance that is also a food additive and for which no food additive regulation has been or can be promulgated. Section 402(a)(2)(A) of the act deems a food adulterated "if it bears or contains any added poisonous or added deleterious substance (other than one which is * * * (ii) a food additive * * *) which is unsafe within the meaning of section 406". If the parenthetical exception had been meant to prohibit issuance of section 406 tolerances for food additives, the exception would have been placed in section 406 of the act. Instead, the language of section 406 of the act allowing the Secretary of Health, Education, and Welfare to promulgate tolerances remains in full effect. Section 406 of the act must, of course, be read as having equal force and effect as the other parts of the statute.

The parenthetical exception was placed in section 402 of the act, the section dealing with the legal definitions of adulteration, rather than in section 406 of the act, the authority to promulgate tolerances, because the intention was to exempt a food additive from the provisions of section 406 of the act once a section 409 regulation has been promulgated. The parenthetical exception is thus similar to the language in sections 406, 408(a), 409(a), and 512(k) of the act granting an exemption from section 402(a)(1) of the act if a regulation under one of those sections is in effect.

The correctness of that interpretation is confirmed by the language of the proviso in section 402(a)(2)(C) of the act. That proviso creates an exemption from the food additive provisions of the act for a pesticide in a processed food under certain circumstances. In doing so, the proviso refers to both sections 406 and 409 of the act. A reference to section 406 of the act would obviously not have been necessary to exempt a substance from the provisions of the act applicable to food additive if the language of section 402(a)(2)(A) of the act made the provisions of section 406 of the act inapplicable to food additives. Instead, because the proviso deals with a situation in which no section 409 regulation will be issued, the reference to section 406 of the act is necessary to afford complete exemption.

Similarly, when a tolerance for an unavoidable pesticide cannot be issued under the criteria of section 408 of the act, section 406 is available to control its use. Under the Executive Order establishing it, the EPA promulgates tolerances under sections 406, 408, and 409 of the act for pesticides in or on food, and the FDA is then responsible for enforcing those provisions.

The Commissioner proposes to establish procedures for controlling all poisonous or deleterious substances. Basic reliance on the provisions of section 409 for regulating use of such substances remains unchanged, and regulations issued under that authority are set forth in 21 CFR Part 121. However, when a food contaminant is unavoidable but cannot be approved under the criteria of section 409 of the act, a formal procedure is being proposed to control its use under authority of sections 306, 402(a), and 406 of the act.

When a tolerance is established under the provisions of section 406, the act requires it to be set at the level necessary for the protection of the public health taking into account the extent to which the substance cannot be avoided and the other ways in which the consumer may be affected by the same or other poisonous or deleterious substances.

At times it will not be appropriate to establish a formal tolerance. The Commissioner is not required to establish a tolerance for every added poisonous or deleterious substance, as is indicated by the language of section 406 of the act recognizing that an adulteration charge can be made under section 402(a)(1) of the act when no tolerance is in effect. Section 306 of the act has long been interpreted to permit the Food and Drug Administration to establish action levels in implementing the adulteration provisions of the act. See United States v. Goodman, 486 F. 2d 847 (7th Cir. 1973); United States v. Thriftimart, Inc., 429 F.2d 1006, 1011 (9th Cir. 1970); United States v. 1500 Cases More or Less, Tomato Paste, 236 F. 2d 208, 211–12 (7th Cir. 1956); United States v. 449 Cases Containing Tomato Paste, 212 F. 2d 567, 572–73 (2d Cir. 1954); United States v. 133 Cases of Tomato Paste, 22 F. Supp. 515, 516 (E.D. Pa. 1938); cf. United States v. Ewig Bros. & Co., supra.

When the factors required to be considered prior to promulgation of a section 406 tolerance are rapidly changing, it would be inappropriate to set such a formal tolerance. The procedures required by section 406 of the act, including a public hearing and requirement of substantial evidence to support the tolerance, contemplate ample evidence to consider, and a relatively stable situation where the evidence will be of more than transient significance and where the tolerance eventually promulgated will be appropriate for a relatively long period of time. For example, if industrial practices are improving so quickly that the extent to which the substance is unavoidable changes significantly from year to year, there is little justification for use of section 406 procedures. Similarly, if toxicological data are scanty or conflicting, but additional data are being developed, it would serve no good purpose to labor over an assessment of the existing data in a public hearing while ignoring the prospects of additional studies. The Commissioner therefore concludes that the structure and criteria of section 406 of the act indicate it is to be used primarily in relatively static circumstances.

When it is not appropriate to promulgate a tolerance under section 406, it may nevertheless still be appropriate to take some formal regulatory action to control use of a substance. In such circumstances, the Commissioner will consider promulgating an action level under authority of sections 306, 402(a), and 406 of the act.

Such action levels are similar to a formal tolerance in basis and effect. In setting an action level, the Commissioner considers evidence indicating when the presence of an added poisonous or deleterious substance may render food injurious to health, which is the standard in section 402(a)(1) of the act. In addition, the Commissioner takes into account the question of its unavoidability, a policy embodied in section 406 of the act. Thus, an action level is based on the same criteria as a tolerance, except that an action level is temporary until the appearance of more stable circumstances makes a formal tolerance appropriate.

Particular problems are associated with respect to the use of pesticides which frequently result in environmental contamination. Tolerances under section 406 of the act may be issued by EPA which allow for residues of pesticides in food, even though the pesticide itself is not registered by EPA for use with that particular crop.

An action level may also be established for a pesticide for which the EPA has not issued a tolerance under section 406 of the act. Because of their joint pesticide responsibilities under the act, the EPA and the FDA agree that both agencies have definite roles in respect to action levels for pesticides. Since action levels are an exercise of enforcement discretion, and since FDA is responsible for enforcement under the Executive Order establishing EPA, FDA is responsible for determining when an action level for a pesticide should be established and issuing such actions levels as regulations. Since action levels are similar to a formal tolerance in basis and effect, and since EPA has the expertise to establish pesticide tolerances, EPA is responsible for determining, upon request from FDA, the appropriate level at which a pesticide action level is to be set.

Such tolerances or action levels properly recognize the problem of environmental contamination caused by drift of a pesticide from one area to another, or the persistence of the chemical in the environment over a period of time. In no instance, however, does any such action level or tolerance itself authorize use of any pesticide on a food crop for which it has not been registered by EPA. Except in the limited instances discussed below, no action level or other administrative action will be taken to permit the sale of food contaminated with a pesticide that has been used in any manner other than in strict accordance with the label directions approved by EPA.

The establishment of a tolerance or an action level does not authorize use of the particular poisonous or deleterious substance involved, or mean that any product containing up to that level of contamination is necessarily a lawful

product. The failure to comply with the requirements of section 402(a)(4) of the act, which prohibits the preparation, packaging, or storage of food under insanitary conditions, is a separate violation of the act, even if the food itself does not violate any tolerance or action level for added poisonous or deleterious substances. Similarly, blending a contaminated food with a noncontaminated food, to produce a final product that falls within a tolerance or action level, is unlawful under the act and is prohibited by the proposed regulations.

The proposed principles and procedures set forth in Part 122 apply equally to imported food. Any food additive or pesticide intentionally used in or on imported food must be the subject of a regulation issued under section 408 or 409 of the act. Unavoidable added poisonous or deleterious substances may be the subject of a tolerance or action level issued under Part 122.

Because of enforcement practicalities, tolerances and action levels for imported food must be the same as for domestically produced food, although levels of unavoidable contamination may differ from country to country. For example, some pesticides are approved for use abroad because of conditions which justify their use, but are not registered for use in this country because those conditions do not exist here. In such a case, a regulation issued by EPA under section 408 or 409 of the act would apply to any food on which the pesticide was intentionally used. A tolerance may be issued by EPA or an action level by FDA, however, to be applied to food unavoidably contaminated by the pesticide. That tolerance or action level will be set at a uniform level for all food, including domestic food. The establishment of the tolerance or action level would not authorize any use of the pesticide in this country, however.

The Commissioner intends to issue, by publication in the FEDERAL REGISTER, all existing action levels for unavoidable poisonous or deleterious substances as regulations in Part 122. In the interim, these publicly known action levels will continue in effect. Although the Commissioner intends to issue as regulations all new action levels as well, circumstances may require use of informal action levels pending issuance of final regulations. Such informal action levels will continue to be announced to the public.

Finally, the Commissioner recognizes that there may be some circumstances where it will be in the public interest to permit the marketing of food that is contaminated with a poisonous or deleterious substance, even though the contamination may have occurred through unlawful or other avoidable misuse of the substance and thus the food would not be eligible for a tolerance or action level. This would occur where a large amount of food is contaminated, resulting in a substantial adverse impact on the national food supply, and all available toxicological data indicate that no significant health hazard is involved. Accordingly, the proposed regulations provide a public procedure whereby, under such circumstances, the Commissioner may permit the marketing of such food, as an exemption from the usual rules that would be applicable. Any such exemptions would be limited to a single occurrence and would be decided on an ad hoc basis after public consideration of the issues involved.

The proposal also prescribes a method of issuing regulations to deal with foods containing naturally occurring poisonous or deleterious substances. Where appropriate, the Commissioner may identify foods which, because of their inherent components, are deemed to be adulterated within the meaning of section 402(a)(1) of the act. These regulations would, of course, not constitute a complete list of such foods.

In the FEDERAL REGISTER of September 23, 1974 (39 FR 34172), the Commissioner consolidated existing regulations on substances prohibited from use in food in a new § 121.106 (21 CFR 121.106). Prohibited food additives will continue to be listed in Part 121, and Part 122 will be used only to identify foods prohibited even when not used as food additives. Since neither of these lists is exhaustive, the appearance of a food or of a poisonous or deleterious substance on one of the lists while not on the other does not constitute a determination that it should not appear on the other.

In addition, the proposal creates a method whereby interested persons can petition the Commissioner to establish a tolerance, action level, or other regulation under this part. Petitions adequately supported would be published in the FEDERAL REGISTER for comment.

No action is proposed for polychlorinated biphenyls (PCB's) (21 CFR 122.10) except recodification as § 122.100. The renumbered section is set forth in its entirety as part of the revision for the convenience of the reader. Additionally, paragraph (a)(9) of that section was stayed by an order published in the FEDERAL REGISTER of August 24, 1973 (38 FR 22792).

Therefore, pursuant to provisions of the Federal Food, Drug, and Cosmetic Act (secs. 306, 402(a), 406, 408, 409, 701(a), 52 Stat. 1045–1046 as amended, 1049, 1055, 68 Stat. 511 as amended; 72 Stat. 1785–1788 as amended; 21 U.S.C. 336, 342(a), 346, 346a, 348, 371(a)) and under authority delegated to him (21 CFR 2.120), the Commissioner proposes that Part 122 be revised to read as follows:

PART 122—POISONOUS OR DELETERIOUS SUBSTANCES IN FOOD AND FOOD-PACKAGING MATERIAL

Subpart A—Definitions and Procedures

Sec.

122.3 Definitions.
122.4 Establishment of regulations.
122.5 Petitions.
122.6 Added poisonous or deleterious substances.
122.7 Unavoidability.
122.8 Exemptions.

Subpart B—Tolerances for Added Poisonous or Deleterious Substances

122.100 Polychlorinated biphenyls (PCB's).

AUTHORITY: Secs. 306, 402(a), 406, 408, 409, 701, 52 Stat. 1045–1046 as amended, 1049, 1055–1056 as amended, 68 Stat. 511, as amended; 72 Stat. 1785–1788 as amended (21 U.S.C. 336, 342(a), 346, 346a, 348, 371).

Subpart A—Definitions and Procedures

§ 122.3 Definitions.

(a) "Act" means the Federal Food, Drug, and Cosmetic Act.

(b) The definitions of terms contained in section 201 of the act are applicable to such terms when used in this part.

(c) A "naturally occurring poisonous or deleterious substance" is a poisonous or deleterious substance that is an inherent natural constituent of a food and is not the result of environmental, agricultural, industrial, or other handling or contamination.

(d) An "added poisonous or deleterious substance" is a poisonous or deleterious substance that is not a naturally occurring poisonous or deleterious substance. When a naturally occurring poisonous or deleterious substance is increased to abnormal levels through mishandling or other intervening act, it is an added poisonous or deleterious substance to the extent of such increase.

(e) "Food" includes human food, substances migrating to food from food-contact articles, pet food, and animal feed.

§ 122.4 Establishment of regulations.

(a) When appropriate under the criteria of § 122.6, a tolerance for an added poisonous or deleterious substance, which may be a food additive, may be established by regulation in Subpart B of this part under the provisions of section 406 of the act. A tolerance may be established at the level of zero.

(b) When appropriate under the criteria of § 122.6, an action level for an added poisonous or deleterious substance, which may be a food additive, may be established by regulation in Subpart C of this part to define the level of contamination at which a food will be deemed to be adulterated under section 402(a)(1) of the act. An action level may prohibit any detectable amount of the substance in food.

(c) A regulation may be established in Subpart D of this part to identify a food containing a naturally occurring poisonous or deleterious substance which will be deemed to be adulterated under section 402(a)(1) of the act. These regulations do not constitute a complete list of such foods.

§ 122.5 Petitions.

The Commissioner of Food and Drugs, either on his own initiative or on behalf of any interested person who has submitted a petition, may issue a proposal to establish, revoke, or amend a regulation under this part. Any such petition shall include an adequate factual basis to support the petition, shall be in the form set forth in § 2.65 of this chapter, and will be published in the FEDERAL REGISTER for comment if it contains reasonable grounds for the proposed regulation.

§ 122.6 Added poisonous or deleterious substances.

(a) Use of an added poisonous or deleterious substance, other than a pesticide chemical, that is also a food additive will be controlled by a regulation issued under section 409 of the act when possible. When such a use cannot be approved under the criteria of section 409, or when the added poisonous or deleterious substance is not a food additive, a tolerance or action level may be established pursuant to the criteria established in paragraphs (b) and (c) of this section. Use of an added poisonous or deleterious substance that is also a pesticide chemical will ordinarily be controlled by a regulation issued under section 406, 408, or 409 by the U.S. Environmental Protection Agency (EPA). When such a regulation has not been issued, an action level for an added poisonous or deleterious substance that is also a pesticide chemical may be established at a level specified by EPA pursuant to the criteria established in paragraph (c) of this section.

(b) A tolerance for an added poisonous or deleterious substance in any food may be established when the following criteria are met:

(1) The substance cannot be avoided by good manufacturing practice.

(2) The tolerance established is sufficient for the protection of the public health, taking into account the extent to which the presence of the substance cannot be avoided and the other ways in which the consumer may be affected by the same or related poisonous or deleterious substances.

(3) No technological or other changes are foreseeable in the near future that might affect the appropriateness of the tolerance established. Examples of changes that might affect the appropriateness of the tolerance include anticipated improvements in good manufacturing practice that would change the extent to which use of the substance is unavoidable and anticipated studies expected to provide significant new toxicological or use data.

(c) An action level for an added poisonous or deleterious substance in any food may be established when the criteria in paragraph (b) of this section are met, except that technological or other changes that might affect the appropriateness of the tolerance are foreseeable in the near future. An action level shall cease to be enforced and shall be revoked when a tolerance for the same substance and use has become effective.

(d) Tolerances will be established under authority appropriate for action levels as well as under authority appropriate for tolerances. In the event the effectiveness of a tolerance is stayed pursuant to section 701(e)(2) of the act by the filing of an objection and request for hearing, the order establishing the tolerance shall be deemed to be an order establishing an action level until final action is taken upon such objection.

§ 122.7 Unavoidability.

(a) Tolerances and action levels in this part are established at levels based on the unavoidability of the poisonous or deleterious substance concerned and do not establish a permissible level of contamination where it is avoidable.

(b) Compliance with tolerances and action levels does not excuse failure to observe either the requirement in section 402(a)(4) of the Federal Food, Drug, and Cosmetic Act that food may not be prepared, packed, or held under insanitary conditions or the other requirements in this chapter that food manufacturers must observe current good manufacturing practices. Evidence obtained through factory inspection indicating such a violation renders the food unlawful, even though the amounts of poisonous or deleterious substances are lower than the currently established tolerances or action levels. The manufacturer of food must at all times utilize quality control procedures which will reduce contamination to the lowest level currently feasible.

(c) The mixing of a food containing a poisonous or deleterious substance in an amount above the tolerance or action level with another lot of food is not permitted and renders the final food unlawful regardless of the amount of poisonous or deleterious substance in the final food.

§ 122.8 Exemptions.

(a) The Commissioner may exempt from regulatory action and permit the marketing of any food that is unlawfully contaminated with a poisonous or deleterious substance if:

(1) He determines (i) based upon all available scientific evidence, that the food is safe for consumption and (ii) that destruction or diversion of the food involved would result in a substantial adverse impact on the national food supply.

(2) Such determination is published as a proposal in the FEDERAL REGISTER, with time for public comment whenever feasible. The time permitted for comment shall depend upon the degree of urgency required by the specific matter involved. In the event that no time for comment is feasible, the exemption may be issued as a final determination rather than as a proposal.

(3) The determination identifies the level of contamination allowed by the exemption, and the food to which it applies (e.g., the specific growing area or other identifying information). The exemption shall not apply to any other food produced in any other area or at any other time. Contaminated food not subject to the exemption will remain subject to regulatory action.

(b) Any person responsible for contamination of the food involved, or shipment of any such food in interstate commerce prior to the issuance of an exemption pursuant to this section, shall remain subject to regulatory action pursuant to sections 302 (injunction proceedings) and 303 (penalties) of the act.

(c) In the case of a poisonous or deleterious substance which is a pesticide chemical, the level of contamination allowed by the exemption shall, upon request of the Food and Drug Administration, be specified by the Environmental Protection Agency pursuant to paragraph (a)(1)(i) of this section and promulgated by the Commissioner. No exemption for a pesticide chemical shall be promulgated without the concurrence of the Environmental Protection Agency.

Subpart B—Tolerances for Added Poisonous or Deleterious Substances

§ 122.100 Polychlorinated biphenyls (PCB's).

(a) Polychlorinated biphenyls (PCB's) are toxic, industrial chemicals. Because of their widespread, uncontrolled industrial applications, PCB's have become a persistent and ubiquitous contaminant in the environment. As a result, certain foods and animal feeds, principally those of animal and marine origin, contain PCB's as unavoidable, environmental contaminants. PCB's are transmitted to the food portion (meat, milk, and eggs) of food producing animals ingesting PCB contaminated animal feed. In addition, a significant percentage of paper food-packaging materials contain PCB's which may migrate to the packaged food. The source of PCB's in paper food-packaging materials is primarily of certain types of carbonless copy paper (containing 3 to 5 percent PCB's) in waste paper stocks used for manufacturing recycled paper. Therefore, temporary tolerances for residues of PCB's as unavoidable environmental or industrial contaminants are established for a sufficient period of time following the effective date of this paragraph to permit the elimination of such contaminants at the earliest practicable time. For the purposes of this paragraph, the term "polychlorinated biphenyls (PCB's)" is applicable to mixtures of chlorinated biphenyl compounds, irrespective of which mixture of PCB's is present as the residue. The temporary tolerances for residues of PCB's are as follows:

(1) 2.5 parts per million in milk (fat basis).

(2) 2.5 parts per million in manufactured dairy products (fat basis).

(3) 5 parts per million in poultry (fat basis).

(4) 0.5 parts per million in eggs.

(5) 0.2 parts per million in finished animal feed for food-producing animals (except the following finished animal feeds: feed concentrates, feed supplements, and feed premixes).

(6) 2 parts per million in animal feed components of animal origin, including fishmeal and other by-products of marine origin and in finished animal feed concentrates, supplements, and premixes intended for food producing animals.

(7) 5 parts per million in fish and shellfish (edible portion). The edible portion of fish excludes head, scales, viscera, and inedible bones.

(8) 0.2 parts per million in infant and junior foods.

(9) 10 parts per million in paper food-packaging material intended for or used with human food, finished animal feed and any components intended for animal feeds. The tolerance shall not apply to

paper food-packaging material separated from the food therein by a functional barrier which is impermeable to migration of PCB's.

(b) A compilation entitled "Analytical Methodology for Polychlorinated Biphenyls, February 1973" for determining compliance with the tolerances established in this section is available from the Hearing Clerk, Department of Health, Education, and Welfare, Room 4–65, 5600 Fishers Lane, Rockville, MD 20852.

Interested persons may, on or before March 6, 1975, file with the Hearing Clerk, Food and Drug Administration, Rm. 4–65, 5600 Fishers Lane, Rockville, MD 20852, written comments (preferably in quintuplicate) regarding this proposal. Received comments may be seen in the above office during working hours, Monday through Friday.

Dated: November 29, 1974.

A. M. SCHMIDT,
Commissioner of Food and Drugs.

[FR Doc.74–28407 Filed 12–5–74;8:45 am]

[21 CFR Part 122]

AFLATOXINS IN SHELLED PEANUTS AND PEANUT PRODUCTS USED AS HUMAN FOODS

Proposed Tolerance

Elsewhere in this issue of the FEDERAL REGISTER, the Commissioner of Food and Drugs is issuing proposed procedural regulations concerning the control of poisonous or deleterious substances in food. In accordance with those proposed procedures and principles, the Commissioner is issuing a proposal for a tolerance for aflatoxins in shelled peanuts and peanut products used as human foods.

Under certain environmental conditions, molds grow on and contaminate some of the foods we eat. Historically, mold contamination of food has been considered as an aesthetic problem; however, there have been numerous instances where consumption of moldy food or feed resulted in illness or death to humans or animals (references 1, 2, 3, and 4). In some of these cases, it was determined that the cause was certain toxic chemical compounds produced by the molds. These compounds are known as mycotoxins (*mykes*, from the Greek, meaning mold). There are many known mycotoxins, but only a few have been found as food contaminants (reference 4).

The recent concern over mycotoxins was brought about by the discovery in the early 1960's of a specific group of mycotoxins known as aflatoxins (reference 1). The aflatoxins are chemically related substances produced by the common molds *Aspergillus flavus* (hence the name aflatoxins, from *A. flavus* toxins) and by *Aspergillus parasiticus*. The term "aflatoxins" also refers to a number of other related chemicals, which either are of synthetic origin or are produced biologically by animals fed aflatoxins. In this document, the terms "aflatoxin" and "aflatoxins" will include only the four common mold-produced aflatoxins known as B_1, B_2, G_1, and G_2.

Aflatoxins may contaminate foods whenever the producing molds grow on foods under favorable conditions of temperature and humidity. The presence of an aflatoxin-producing mold on a food does not necessarily imply the presence of the aflatoxins. Conversely, the absence of obvious growth of an aflatoxin-producing mold does not mean the absence of the toxins, since aflatoxins may be produced when there is little evident mold growth. Furthermore, aflatoxins may remain in a food product after processing has obscured or otherwise eliminated visible mold (references 3 and 4). Under laboratory conditions, a large number of foods can support the growth of aflatoxin-producing molds; under natural conditions in the field, natural competition determines which molds will grow and which mycotoxins will be produced. Corn, barley, copra, cassava, tree nuts, cottonseed, peanuts, rice, wheat, and grain sorghum are subject to natural aflatoxin contamination (reference 4). In the United States, aflatoxins have been detected only in corn, figs, grain sorghum, cottonseed, certain tree nuts, and peanuts (reference 5).

Aflatoxins are present in peanuts and peanut products because of these contaminating molds. They are, therefore, added substances within the meaning of the Federal Food, Drug, and Cosmetic Act. The meaning of that term is discussed in detail in the preamble to the proposed procedures and principles for the control of poisonous and deleterious substances published elsewhere in this issue of the FEDERAL REGISTER.

The Commissioner has a two-fold concern with aflatoxins. First, animals fed aflatoxins are subject to acute toxic effects. Second, and of primary concern, data which indicate that these substances, particularly aflatoxin B_1, found in some species of test animals are among the most potent liver carcinogens known (references 1, 2, and 3).

The acute toxic effects of the aflatoxins are of little concern in the United States, since our agricultural and marketing processes assure that heavily molded food, which might contain levels of aflatoxins associated with such effects, does not enter the human food chain. Of great concern is the effect of long-term, low-level ingestion (i.e., chronic toxicity effects), particularly the carcinogenic effects (references 6, 7, and 8).

While there is no direct evidence that aflatoxins are a cause of liver cancer in man in the United States, their extreme potency as hepatocarcinogens in some animal species does furnish a foundation for the Commissioner's concern. The carcinogenicity of aflatoxins has been demonstrated in rats, monkeys, rainbow trout, ducks, ferrets, and mice (references 6, 7, and 8).

Aflatoxin carcinogenesis has been studied most extensively in rats. There have been several experimental studies with rats, and liver carcinomas have been produced in a number of different strains (reference 7).

In one study, highly purified aflatoxin B_1 was fed continuously to 25 rats for a period of 68–80 weeks. The level of aflatoxin in the diet was 15 parts per billion (ppb). All 25 rats developed liver carcinoma (reference 9). In a later study with the same strain of rat, liver carcinoma was produced in 2 out of 22 rats exposed for 105 weeks to a diet containing only 1 ppb aflatoxin B_1 (reference 9). These two studies demonstrate the extreme potency of aflatoxin B_1 as a liver carcinogen; however, other experimental studies furnish evidence that there are marked differences in the incidence of liver carcinomas in rats produced by various levels of aflatoxins depending on such variables as composition of the diet, age of the test animals, strain of rats used, and length and type of exposure (reference 7). The extreme difficulty in drawing conclusions from all of these studies is indicated by one study in which one strain of rat exposed to aflatoxins at a level of 80 ppb for 90 weeks suffered some liver damage, but no cancer (reference 10).

Liver cancer has been produced by ingestion of aflatoxins in three Macaque monkeys. One was exposed to an average of 0.02 milligram per kilogram (mg/kg) body weight daily for 5½ years. The other two were exposed to an average of 0.06 mg/kg body weight for 6 years (reference 8).

Epidemiological studies bearing on the possible effects of aflatoxin in man have been performed on specific population groups in Southeast Asia and Africa, where there is a known high incidence of primary liver cancer (references 8 and 11). In these areas, the climate and agronomic practices were expected to be conducive to production of aflatoxins in food. In one of the areas studied, 95 percent of the corn and 80 percent of the peanut samples had average aflatoxin levels around 100 ppb. In another area, 20 percent of the food staples contained aflatoxin levels averaging 300 ppb. In a third area, there was a high incidence (7 percent) of low levels (3–4 ppb) of aflatoxins in family meals. The result of these studies is an indication of a general correlation between the incidence of primary liver cancer in humans and the exposure to aflatoxins (reference 11).

No epidemiological studies of these types have been carried out in the United States. However, primary liver cancer is a minor form of cancer in the United States, accounting for about 0.7 percent of all cancer cases (reference 12). In addition, cancer rates in those areas of the United States where climatic conditions are most conducive to aflatoxin contamination (i.e., the southeastern states) do not differ significantly from other areas (reference 13). Domestic profiles for primary liver cancer differ in sex and age from those observed in "high incidence" countries (reference 13). One study in a major city in the United States shows some degree of correlation between primary liver cancer and alcoholism (reference 14).

Thus, while it is not certain that aflatoxins are a cause of primary liver cancer in the United States, the Commissioner concludes that the observations of severe carcinogenic effects in experimental animals and positive correlations between dietary aflatoxins and primary human liver cancer seen in other parts of the world are sufficient justification to regard aflatoxins as poisonous or deleterious substances and to take actions to hold the human exposure to aflatoxins in the United States to the lowest level possible. Shortly after the discovery of the presence of aflatoxins in peanuts (reference 15), the Food and Drug Administration (FDA) began a program for their control (references 5 and 16). In 1965, the FDA established an informal action level of 30 ppb total aflatoxins. In 1969, it was reduced to the current level of 20 ppb total aflatoxins.

The informal action level of 20 ppb has been applied to all affected commodities. As more is learned about the extent to which contamination of foods by aflatoxins can be avoided, FDA will systematically and formally adjust its action level to reflect this new knowledge either as an action level or tolerance. Since different agricultural products become contaminated in different ways and since the potential for removing contamination during processing differs from one product to another, the extent to which aflatoxin contamination can be avoided is commodity dependent. Action levels or tolerances will be established, then, on a commodity-by-commodity basis. In each case, the action level or tolerance will be consistent with the concept that the total human exposure to aflatoxins must be as low as possible.

At the present, there exists a body of information upon which to judge the extent to which aflatoxin contamination can be avoided in shelled peanuts and peanut products used as human food, and a reconsideration of the action level for these commodities is in order. FDA is in the process of collecting relevant information on other affected commodities. When this information becomes adequate in scope and content, adjustments in the acceptable level will be made for each of the other affected products. Until then, the 20 ppb action level will be maintained.

It is now known that aflatoxins can contaminate peanuts during any of several stages: growth, harvest, shipment, or storage. Whether mold develops at any or all of these stages depends on such factors as kernel damage, temperature, moisture, and availability of oxygen (references 17 and 18).

Although aflatoxin contaminated peanuts can be found prior to harvest, the major point of contamination, by all available evidence, is the curing process. Once mold invasion takes place, aflatoxins may be produced unless the moisture content is rapidly reduced to a level unfavorable to mold growth (references 17, 18, and 19).

Employed properly, artificial drying is an effective way to control contamination; nevertheless, large quantities of peanuts are still dried naturally, thereby risking contamination due to unfavorable weather conditions (reference 17). Fuel shortages are apt to increase the problem, since artificial drying may give way to natural, open air drying practices.

Damage to the peanut shell may be one of the most significant factors leading to mold growth and aflatoxin contamination. It has been demonstrated that the removal of damaged kernels can lead to a major reduction in the aflatoxin levels in peanuts (references 15 and 19). Insect infestation is one source of damage; however, many kernels are damaged as the result of mechanical harvesting techniques (reference 17). Once the natural barrier imposed by the shell is breached, mold invasion can begin.

Fungicides, crop rotational practices, and other good agronomic practices can ameliorate the problem to an extent (reference 17). It is impossible at this time totally to eliminate all of the breakdowns that can occur in the total process of growing, harvesting and storing peanuts. Adverse weather, insects, improper harvesting methods, and general ignorance of the problems and solutions will be with us for some time to come. Thus, in practice, the complete prevention of mold growth and aflatoxin production in peanuts appears unattainable at the present time.

Processors of shelled nuts can eliminate some contaminated material by using a number of standard manual, mechanical, and electronic sorting procedures. Although these procedures were originally designed to remove the damaged, discolored, and immature nuts which affect appearance and flavor, they have been useful in removing aflatoxin-containing peanuts, since contamination is often associated with such peanuts (references 15, 20, and 21). Sorting does have a major limitation in that intact, sound, mature kernels can contain aflatoxins (reference 22). Due to this and other as yet unidentified reasons, there is a limit, not easily definable, to the effectiveness of sorting techniques.

The ordinary process of roasting peanuts results in some reduction of average aflatoxin level, but complete elimination of aflatoxins cannot be accomplished by the process (reference 23).

In order to detect the presence of aflatoxins in peanuts, it is necessary to have analytical methods of adequate sensitivity and specificity. Such methods are available and their effectiveness has been demonstrated by interlaboratory collaborative studies. These are now official methods of the Association of Official Analytical Chemists (AOAC) (references 3, 24, and 25). AOAC Method I can be used to detect aflatoxins B_1, B_2, G_1, and G_2 in peanut products at levels as low as 1–2 ppb (total aflatoxins). AOAC Method III, although not as accurate as Method I at low levels (2–10 ppb), is much more rapid and is used as an industrial quality control method (references 25, 26, and 27). Tests for confirmation of identity of measured aflatoxins B_1 and G_1 have also been studied collaboratively and are official AOAC (reference 24).

While the analytical methods are a source of variability in the assay for aflatoxins, the greatest source of variability is sampling. Contamination is usually confined to a small number of kernels and there is thus no practical sampling plan which can assure that a sample is truly representative of the entire lot under investigation. Therefore, any practical sampling plan must be based on certain distribution assumptions and can assure only a limited level of confidence that a particular sample is representative of a lot (reference 28).

FDA regulation of finished peanut products has been only a part of the total control picture. The U.S. Department of Agriculture (USDA), through its Peanut Marketing Agreements, has been involved in establishing and maintaining sampling and analysis procedures designed to remove lots of peanuts showing visible *A. flavus* mold and lots of aflatoxin-contaminated raw peanuts prior to finished product manufacture (references 16 and 29). FDA has been involved in this program on a consulting basis and receives yearly, for analysis and evaluation, the USDA reports of analyses of raw, shelled peanuts (reference 30). This program requires that all shelled peanuts offered for sale to manufacturers or wholesalers be sampled and analyzed for aflatoxins by a laboratory certified as competent by the Peanut Administrative Committee (references 5 and 21). A negative certification means that the sample assay is not over 25 ppb total aflatoxins (reference 21).

Once a lot of peanuts is sampled and assayed, it may or may not be tested again as practices vary among manufacturers. Some manufacturers do maintain their own in-house laboratories, or contract for testing services through independent or government laboratories. Others perform no further testing at all, relying on the processing procedure to remove the aflatoxins.

Since the inception of this system of controls, sampling and analysis by FDA has turned up few violative samples of finished peanut products (reference 5). FDA surveillance, however, has been limited. In 1973, the agency conducted its first large survey of the nation's peanut products. This was done to determine the nature of the quality control procedures being utilized by manufacturers to remove contaminated peanuts and the effectiveness of these procedures (reference 31).

The survey was also used to determine the distribution of various levels of aflatoxins in the nation's peanut product supply. These survey data are used as one of the considerations in establishing a tolerance for aflatoxins in peanut butter and other peanut products. All analytical data gathered in surveys of this type and reported here include the errors associated with sampling and analysis of aflatoxins.

The FDA survey encompassed 146 peanut butter and/or peanut product

manufacturers. Of prime concern was geographical and size distribution, with the greater emphasis on smaller concerns where the poorest operating procedures and quality control were presumed to exist. A checklist was provided to each FDA inspector to ensure uniform reporting and data retrieval. From the 146 establishments, 361 samples of peanut butter and other finished peanut products (generally unground) were collected.

Aflatoxins levels above the action level (total aflatoxins, 20 ppb) were found in 4 percent of the 141 peanut butter samples and 2 percent of the 220 samples of unground peanuts. These samples with levels above the action level came from 5 percent of the establishments inspected. Detectable aflatoxins (greater than approximately 1 ppb) were found in 25 percent of the peanut butter samples and 10 percent of the unground peanut samples. These samples came from 22 percent of the establishments inspected. There is a large error inherent in sampling unground nuts for aflatoxins compared to the blended product, peanut butter. This precluded drawing a statistically valid inference that the incidence of aflatoxins in unground nuts is lower than that in peanut butter.

The data were examined for possible correlations between aflatoxin findings and size of establishment, between aflatoxin findings and manufacturing practices related to control of aflatoxin contamination, and between aflatoxin findings and the aflatoxin level in the raw peanuts used.

Aflatoxin levels above 20 ppb were found only in the small and medium size establishments; however, 50 percent of the 10 largest establishments provided samples with detectable aflatoxin, compared to 23 percent of the 136 small and medium size establishments (reference 31). No correlation between establishment size and incidence and degree of contamination could be found for the establishments inspected.

In the instances where aflatoxin contamination was found in the consumer product, there was no obvious correlation between the levels of aflatoxin found and the various manufacturing practices used (reference 31). The same observation holds for both peanut butter and unground peanuts.

There were indications that the processing of peanuts could result in a reduction of aflatoxin level to a trace of nondetectable level. However, the level of aflatoxin in raw peanuts was not consistently related to the level in the consumer product. In 15 instances, the level in the consumer product was higher than that found in the raw peanuts. This result emphasizes the difficulty in obtaining a representative sample of the granular product (raw peanuts), as aflatoxin contamination may be extremely heterogeneous within a lot (reference 31).

The presence of detectable levels of aflatoxins in consumer products could result either from faulty application of the reported inspection and sorting practices or a peculiarity in the nature and distribution of the contamination. The latter possibility has some justification since aflatoxin has been found in otherwise sound, undamaged kernels.

One important conclusion which derives from all of these findings is that current manufacturing capabilities cannot consistently assure the complete elimination of aflatoxin-contaminated peanuts from a lot during processing. What level can be consistently reached is determined by a detailed consideration of the FDA and other survey data, including those gathered by the USDA analyses of peanut butter produced under government contract and those collected by the Canadian Health Protection Branch (reference 30).

Thus, in determining an acceptable level for aflatoxins in shelled peanuts and other peanut products, the Commissioner considered all of the available information and sought to bring into balance four factors:

1. The need to minimize human exposure to aflatoxins;

2. The capabilities of sampling procedures and analytical methods to detect, measure and confirm the aflatoxins;

3. The capability of agricultural and manufacturing technology to prevent and remove contaminated peanuts; and

4. The need for the continued availability of a low cost protein source (i.e., peanuts (reference 32)).

Survey data (FDA, USDA, and Canadian) indicate that since 1971 an average of 93 percent of sampled peanut products contained aflatoxins below the 20 ppb level (references 30 and 31). The Commissioner concludes that current agricultural and manufacturing technology is capable of meeting a level below that which it is now being asked to meet. Although the immediate result of lowering the level may be the loss of a small percentage of the peanut supply, improved producer practices should compensate for any short-run loss.

Obviously, for complete protection, aflatoxins should be eliminated from food, but this is not presently feasible. Therefore, it is necessary for the Commissioner to weigh the consequences of possible levels above zero.

Setting a level at 5 or 10 ppb was considered by the Commissioner. Eleven of the 12 major U.S. manufacturers of peanut butter could meet a 10 ppb level in 90 percent of their products (reference 33). The Canadian government survey of 1972 indicated that 95 percent of the samples of peanut butter could meet this standard. However, there is year-to-year variability in aflatoxin contamination of the peanut crop. The FDA survey of 1973 evidenced the fact that only 89 percent of the peanut butter samples and 82 percent of the establishments surveyed met a 10 ppb level. Of the 12 largest establishments surveyed, only seven met a 5 ppb limit in 90 percent of their samples (reference 33). There are also data showing that in some years only about 60 percent of the peanut butter produced met a 5 ppb level (reference 30). One bad crop year could effectively eliminate a large percentage of peanut products from the market. Thus, a move to 5 or 10 ppb could result in significant losses to producers, manufacturers, and consumers alike.

These increased losses of food would result in much higher prices (reference 33) or in unavailability of what is generally considered a highly nutritious and useful food.

Another important consideration concerns setting a tolerance at a level at which manufacturers have the capability to monitor their products during processing and to ensure that the finished product complies with the tolerance. Manufacturers of peanut products now monitoring their production set analytical limits for their products in the range 5 to 10 ppb to ensure that their finished products do not exceed the current 20 ppb action level; this factor must be built into a quality control operation because of the large error associated with sampling and analysis. Setting a tolerance level of 5 or 10 ppb for aflatoxins in peanut products would have the effect of requiring manufacturers to employ an analytical limit of less than 1 to 5 ppb for quality control purposes. Because present sampling and analytical methodologies have considerable error at the 1–5 ppb range, analytical results obtained for such quality control samples would not accurately represent the production lot from which the sample was drawn. Therefore, the capability of manufacturers to control their production at these levels is extremely questionable, and to guard against the release of products containing levels of aflatoxins in excess of 5 or 10 ppb is not possible.

It would serve no useful purpose to establish a tolerance at a level at which current sampling and analytical procedures are such that compliance with the tolerance is unachievable.

In addition, because there is no direct evidence that aflatoxins cause cancer in man or of what may be the level of no effect, the Commissioner cannot conclude that there is any tangible gain from lowering the permissible level to either 10 or 5 ppb. Such uncertain benefit to the public health must be weighed against the clear loss of food that would result.

On the basis of these considerations, the Commissioner concludes that justification does not exist for implementing a maximum permissible level at either 10 or 5 ppb.

A level of 15 ppb represents a lowering of the level from the 20 ppb level which has stood since 1969. FDA survey data indicate that 93 percent of the lots of peanut butter and 97 percent of the lots of other peanut products could meet the 15 ppb level (reference 31).

Survey data from the Canadian Government and from USDA show that, on the average, 97 percent of the lots of peanut butter sampled since 1971 could meet the proposed level (reference 30).

The Commissioner concludes that existing agricultural and industrial technology can combine to yield finished peanut products at or below a 15 ppb level

without causing significantly increased losses of food. This level will also allow manufacturers to maintain capability for monitoring their products during processing with nearly as much reliability as could be attained with a 20 ppb level. At the same time, the lowering of the allowable level to 15 ppb will result in decreased human exposure to aflatoxins.

In setting a tolerance or action level, the Commissioner considers the other ways in which the consumer may be affected by the same substance or by other substances. Since there is no known acceptable level of intake of aflatoxins and the proposed tolerance is set at the lowest practicable level, the existence of other sources of aflatoxins and the existence of other poisonous or deleterious substances do not affect the level proposed for aflatoxin in shelled peanuts and peanut production.

The regulation also prescribes sampling and analytical methods by which compliance with the proposed limitation will be determined. Because of possible variation in the results obtained by the analytical method prescribed, the regulation provides that the average of duplicate analyses shall be used.

Taking into account (1) the fact that aflatoxins cannot be avoided by current agronomic and manufacturing processes; (2) the extent to which aflatoxins are unavoidable; (3) the data on the effects of aflatoxins; and (4) the other ways in which the consumer may be affected by aflatoxins and other substances, the Commissioner proposes to establish a tolerance for aflatoxins in shelled peanuts and peanut products used as human foods, above which such products will be deemed unsafe within the meaning of section 406 of the act. Accordingly a tolerance of 15 ppb is proposed.

Pending a final regulation, FDA will continue to apply for enforcement purposes an action level of 20 ppb for aflatoxin in shelled peanuts and peanut products.

The Commissioner has considered promulgating the present proposal as an action level. Limiting an unavoidable substance through use of an action level, rather than a tolerance, is appropriate when changes are foreseeable in the near future that might affect the appropriateness of the limitation established. This, however, is not the case with aflatoxins. The Commissioner is unaware of any new or anticipated developments that would affect the proposed 15 ppb level in the near future. There are no new procedures pending, either agronomic or technical, that will alter the present unavoidability of aflatoxins. Futhermore, it is unlikely that any new pertinent information will become available in the near future. Therefore, it is the Commissioner's preliminary conclusion that the most reasonable approach to regulating aflatoxin, in shelled peanuts and peanut products, would be a formal tolerance established under section 406 of the act. The Commissioner, however, specifically solicits comments on the question of whether any anticipated changes make an action level more appropriate.

References

A copy of each reference document is on file with the Hearing Clerk, Food and Drug Administration, Rm. 4–65, 5600 Fishers Lane, Rockville, MD 20852, and may be seen during working hours Monday through Friday.

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Therefore, pursuant to provisions of the Federal Food, Drug, and Cosmetic Act (secs. 306, 402, 406, 701, 52 Stat. 1045–1046 as amended, 1049, 1055–1056 as amended by 70 Stat. 919 and 72 Stat. 948; 21 U.S.C. 336, 342, 346, 371) and under authority delegated to him (21 CFR 2.120), the Commissioner proposes that Part 122 be amended by adding a new § 122.101 as follows:

§ 122.101 Aflatoxin in shelled peanuts and peanut products used as human food.

(a) A tolerance of 15 parts per billion (ppb) is established for total aflatoxins (B_1, B_2, G_1, G_2) in shelled peanuts and peanut products used as human food.

(b) Compliance with this section shall be determined as follows:

(1) A lot shall consist of a collection of primary containers or units of the same size, type, and style produced under conditions as nearly uniform as possible, designated by a common container code or marking, or in the absence of any common container code or marking, a day's production.

(2) (i) The sample for aflatoxin analysis of smooth peanut butter shall consist of a composition of 24 subsamples

(primary units) randomly drawn from a lot.

(ii) For all other peanut products, the sample shall consist of a composite of 48 subsamples (primary units) randomly chosen from a lot, or if from a bulk lot, 48 1-pound subsamples.

(3) Composites shall be analyzed in duplicate using 50-gram portions from the lot sample by the methods of the Association of Official Analytical Chemists (AOAC), as described in "Official Methods of Analysis of the Association of Official Analytical Chemists," 11th Ed., Chapter 26, Secs. 26.015–26.020.[1]

(4) A lot does not comply with the tolerance established in paragraph (a) of this section when:

(i) The average value of the two analyses of the composite of randomly chosen subsamples is greater than 15 ppb total aflatoxins; and

(ii) The identity of aflatoxins in the analytical sample is confirmed by chemical derivative formation (AOAC, Official Methods of Analysis, 11th Ed., Chapter 26, Secs. 26.054–26.055).

Interested persons may, on or before March 6, 1975, file with the Hearing Clerk, Food and Drug Administration, Rm. 4–65, 5600 Fishers Lane, Rockville, MD 20852, written comments (preferably in quintuplicate) regarding this proposal. Received comments may be seen in the above office during working hours, Monday through Friday.

Dated: November 29, 1974.

A. M. SCHMIDT,
Commissioner of Food and Drugs.

[FR Doc.74–28408 Filed 12–5–74;8:45 am]

[1] Copies may be obtained from: Association of Official Analytical Chemists, P.O. Box 540, Benjamin Franklin Station, Washington, D.C. 20044.